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LETTERS TO THE EDITOR

Transient Left Ventricular Apical Ballooning Without Coronary Artery Stenosis: A Form of Stunning-Like Phenomenon?

We read with great interest the study published by Tsuchihashi et al. (1) in the July issue of the *Journal*. The study presented detailed findings with regard to the unusual phenomenon that mimic acute coronary syndrome.

Left ventricular (LV) contraction abnormalities without coronary stenosis have been previously reported in physically or emotionally stressed patients (2–4). Epicardial coronary spasm has not been confirmed in these patients even while ST-segment is elevated in the electrocardiogram (ECG). Kawai et al. (3) stated this could be a form of cardiomyopathy, termed "ampulla cardiomyopathy". However, Tsuchihashi et al. (1) suggested that the histological change was similar to that of catecholamine-induced myocardial damage and that microvascular spasm was involved.

We have been interested in this form of reversible LV dysfunction, and recently reported the involvement of impaired coronary microcirculation in transient LV contraction abnormalities (5). Coronary arteriography revealed no significant stenosis in the epicardial arteries. Relative coronary flow reserve measured by intracoronary Doppler guide wire was significantly reduced, which suggested severely decreased coronary microcirculation in these patients. Contrast myocardial echocardiography revealed that the impaired ventricular perfusion was reversible. According to our findings about coronary microcirculation, the histological changes seen in these patients might be a result of a stunning-like phenomenon due to microvascular abnormalities.

Finally, the investigators carefully used the term "transient left ventricular apical ballooning" in the title of their study (1). The exact mechanism of this reversible contraction abnormality still remains unclear. A term defining the pathogenesis of this syndrome will be necessary in the near future.

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REPLY

We appreciate the interest shown and the comment given by Ako et al. in regard to our recent clinical study (1) on a heart syndrome with transient left ventricular (LV) apical ballooning without coronary artery stenosis mimicking acute myocardial infarction. As mentioned in my study and others (2,3), coronary vasospasm under various physical and mental stresses, including administration of adrenergic drugs, might be considered as an initial etiological basis of this novel syndrome. Impaired coronary microcirculation in this syndrome was shown by Ako et al. (4) using an intracoronary Doppler flow-wire technique. The possibility of transient ischemia including microvessel vasospasm as an initiating factor of this syndrome could not be ruled out; however, we speculate that vasospasm is not the main cause, for the following several reasons. First, autopsy findings in some cases were different from those of myocardial ischemia (5). Second, impaired microcirculation during the follow-up period will not be direct evidence for considering the etiology of this syndrome, because the possibility of delayed recovery of impaired microcirculation due to transient wall motion abnormality is not excluded in this syndrome. Recent scintigraphic evaluation by Dr. Owa (co-author) showed a transient (but persistent for several months) perfusion–metabolism mismatch in the apex (6). Our study also showed a representative case with delayed recovery of coronary microcirculation (1).

Important etiological causes suspected from our study include stress cardiomyopathy caused by vigorous stress (cathecholamine exposure) (6–8), dynamic midventricular obstruction due to basal hypercontraction (9) and/or secondary myocardial ischemia caused by apical ballooning (increased wall tension). However, as already mentioned in the discussion (1), our study was a retrospective investigation and there are several limitations. Further cases, therefore, should be investigated to determine the pathogenesis of this heart syndrome.

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